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Project Summary

Effects of 2% Venous Carboxyhemoglobin on Exercise-Induced Angina Pectoris

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This study is the latest in a long series examining the effects of exposure to carbon monoxide from smoking or heavy atmospheric pollution on cardiovascular function. It attempts to determine whether a venous carboxyhemoglobin level of 2% does or does not aggravate exercise-induced angina pectoris.

In a double-blind, randomized, crossover study, 15 patients with angina pectoris breathed 50 ppm of carbon monoxide for 1 hour which raised the mean venous carboxyhemoglobin from 1.09 to 2.02%. They also experienced a reduction in mean exercise time until the onset of angina pectoris from 321.7 to 289.2 seconds, $P < 0.001$. After breathing compressed, purified air to reduce the mean venous carboxyhemoglobin from 1.07 to 1.00%, there was an insignificant increase in mean exercise time until the onset of angina pectoris from 324.5 to 330.3 seconds. In comparison with the control period, or after breathing compressed, purified air, a 2.02% venous carboxyhemoglobin level caused a reduction in exercise time until the onset of angina pectoris, $P < 0.001$. No significant change occurred in resting systolic blood pressure times heart rate; a reduction was noted in systolic blood pressure times heart rate at angina pectoris, $P < 0.001$. No significant change occurred in exercise-induced ischemic ST-segment depression. These data indicate that a 2% venous carboxyhemoglobin level aggravates angina pectoris.

This Project Summary was developed by EPA's Health Effects Research Laboratory, Research Triangle Park, NC 27711, to announce key findings of the research project that is fully documented in a separate report of the same title (see Project Report ordering information at back).

Introduction

Exposure to carbon monoxide from smoking or heavy atmospheric carbon monoxide pollution impairs cardiovascular function in patients with cardiovascular disease, in patients with chronic obstructive pulmonary disease, and in normal persons. This impaired function is manifested as a reduction in exercise time until the onset of angina pectoris in patients with coronary heart disease. It also causes ischemic ST-segment depression ≥ 1.0 mm (to occur earlier and after less exertion) after exercise-induced angina pectoris.

Previous studies have confirmed that venous carboxyhemoglobin levels of 2.5%-3.0% aggravate angina pectoris secondary to coronary heart disease. The purpose of the current study is to determine in a similar study design

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whether a venous carboxyhemoglobin level of 2% does or does not aggravate exercise-induced angina pectoris.

Materials and Methods

Fifteen patients, 14 men and one woman, mean age 59.0 ± 7.2 years, with stable angina pectoris due to angiographically documented coronary artery disease were subjects. Eight of the 15 patients (53%) also had a previously documented healed myocardial infarction. None of the 15 patients were smokers or had anemia.

On two successive study mornings, at 8 a.m., with the subject in the fasting state, venous blood was drawn and analyzed for carboxyhemoglobin and hemoglobin levels (and for methemoglobin levels in the last eight patients) with an IL 282 Co-Oximeter.

Simultaneous leads II and V_5 were then recorded with an electrocardiograph with the patient sitting on the bicycle ergometer. The resting heart rate was obtained from this electrocardiogram. The resting blood pressure was measured with a mercury sphygmomanometer. Each subject then exercised upright on the bicycle ergometer with a progressive work load until the onset of anginal discomfort. The work load initially was 50 watts and increased 25 watts every three minutes. The subject's electrocardiogram was monitored throughout exercise and was recorded at the onset of angina pectoris. The blood pressure was recorded at the onset of angina pectoris with the patient continuing to exercise until the blood pressure was recorded.

In a double-blind, randomized, crossover study, each subject breathed 55 ppm of carbon monoxide for 1 hour on one morning and compressed, purified air for 1 hour on the other morning. Eight of 15 patients breathed carbon monoxide on day 1 and compressed, purified air on day 2, with the schedule for the other seven patients being the reverse.

After the patient breathed carbon monoxide or compressed, purified air for 1 hour, venous blood was again drawn and analyzed. Electrocardiograms were again taken at rest and after the onset of anginal discomfort during exercise.

The electrocardiograms were analyzed in a blind manner after the study was completed. The data were analyzed with Student's t-test for correlated means.

Conclusions and Recommendations

These data show that breathing carbon monoxide to raise the mean venous carboxyhemoglobin level from 1.09% to 2.02% in comparison to the control period or in comparison to breathing compressed, purified air does not affect the resting product of systolic blood pressure times heart beat. It does, however, reduce the product of systolic blood pressure times heart rate at the onset of angina pectoris, as well as reducing the exercise duration until the onset of anginal pain.

The significance of these data is that a 2% venous carboxyhemoglobin level aggravates angina pectoris. This information should be considered in setting the national air quality criteria for carbon monoxide, since it indicates that a safe threshold of carbon monoxide exposure for patients with angina pectoris due to coronary artery disease would have to be very low.

A 10% reduction in exercise tolerance until the onset of angina pectoris at 2% venous carboxyhemoglobin level is clinically important and may precipitate anginal pain during activities which could be performed without developing anginal pain at a 1% venous carboxyhemoglobin level.

Patients with unstable angina pectoris or acute myocardial infarction are more susceptible than patients with stable angina pectoris to adverse effects from low levels of carbon monoxide exposure. The more critical the coronary artery disease, the more susceptible is the individual to adverse health effects from low levels of carbon monoxide exposure. Low levels of carbon monoxide exposure may contribute to myocardial infarction and to death in these persons. It should also be taken into consideration that persons who have had no prior recognized symptoms of heart disease make up 25% of all sudden deaths from coronary heart disease.

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***George M. Goldstein** is the EPA Project Officer (see below).*

The complete report, entitled "Effects of 2% Venous Carboxyhemoglobin on Exercise-Induced Angina Pectoris," (Order No. PB 81-152 480; Cost: $6.50, subject to change) will be available only from:

National Technical Information Service
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